THE

VETERINARY JOURNAL

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MARCH, 1939

Editorial

THE VETERINARY PROFESSION AND ITS RELATION TO THE POULTRY INDUSTRY

IT becomes more and more apparent that the Poultry Industry in this country is not in the flourishing condition necessary for its well-being and that drastic steps are necessary to ensure that it shall be put on a sound economic basis. The Report issued in 1937 by the Poultry Technical Committee for Great Britain and Ireland emphasised the part which disease has played in the production of the present situation and we are pleased to note that the Minister of Agriculture has stated definitely within very recent times that it is the intention of the Government to introduce this Session " legislation for promoting the welfare of the poultry industry " which will deal with " the whole of the industry throughout the country." We await with great interest the publication of the Government's proposals.

The Veterinary Profession will certainly be required to play a considerable part in any scheme which may be brought into operation, for it is realised

everywhere that veterinary surgeons must, by their special training, be the only competent group of people who can deal adequately with problems of disease and methods of their control. We, as a profession, have probably failed to interest ourselves to a marked degree in poultry problems in the past. It must always be remembered, however, that a number of laboratory workers and some practitioners have studied disease and other problems and have added very materially to our general and specific knowledge on the subject. It is not too much to claim that practically all our methods of controlling infectious diseases in poultry are the direct outcome of work carried out by members of the Veterinary Profession. On the other hand, however, there has been in the past some lack of interest displayed by the Profession as a whole in the poultry keeper's problems. We know now, of course, how many of these problems have arisen and how much of the cause must be attributed to the Industry itself in failing to realise how seriously indiscriminate breeding can influence the health of their stocks. The recommendations of the Poultry Technical Committee, if adopted by the Government, should go a long way in the course of a few years to reducing the incidence of disease and placing the health of poultry generally on a sounder basis. The Veterinary Surgeon must play his part in any scheme which is evolved and it is of much interest to be able to record that more and more encouragement and opportunity is being given to the profession to become acquainted with the problems of disease and health in poultry stock throughout the country.

This Journal has, in the past, devoted special issues to poultry subjects and has kept before its readers the necessity for encouragement in pursuing the study of disease and other poultry problems by veterinary research workers. It is to be hoped that full use will be made of the profession—practitioners as well as laboratory workers — in national schemes for the control of the health of poultry; and that the scheme of research proposed by the Poultry Technical Committee will materialise so that members of the Veterinary Profession will be brought into the scheme in all its aspects. The profession, too, should be consulted in regard to the working of the schemes.

We can assure the Minister and his colleagues that he will have our loyal support in any scheme which the Government brings into being.

General Articles

LYMPHOMATOSIS OF TURKEYS

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DURING the past few years, in the course of routine post-mortem examinations upon a very large number of turkeys, the writer has come across cases where the autopsy has revealed collections of cells of the lymphoid series which many pathological workers have been pleased to regard as evidence of "true fowl paralysis," more aptly called lymphomatosis. These lesions have been found in the liver and cæca of birds which had suffered from blackhead or entero-hepatitis.

Three outbreaks are worth recording, because they may throw some light upon the pathological changes which take place in chickens suffering from lymphomatosis.

The five constant factors in these outbreaks were:

(1) The very high mortality which occurred, namely, 87, 62.5 and 79 per cent. respectively.

(2) The large number of blackhead lesions found at post-mortem examination of the birds which died in these outbreaks.

(3) The distribution of the lymphomatous lesions in the usual sites of those of blackhead.

(4) The fact that other birds had died and shown typical uncomplicated blackhead lesions on post-mortem examination, shortly before those showing lymphomatosis.

(5) The fact that the birds were kept on a small piece of land which had for many years previously been used for turkey and chicken rearing.

It has been my experience that young turkeys are particularly susceptible to most diseases of the domestic fowl, and unless environmental conditions are good there is a high mortality.

These observations certainly apply to blackhead, to which disease other species of poultry have a relatively high degree of immunity. On account of this disease it is impossible to rear turkeys upon most poultry farms, unless ground not previously used for poultry is available for them.

During the first few weeks of their life turkeys are particularly susceptible

to chilling, draughts and variations of temperature, and heavy losses occur from these causes. If lymphomatosis is a specific infectious disease affecting birds during their early life, as some workers think, we should surely have very many more outbreaks reported in turkeys.

The accumulations of lymphoid cells found in these outbreaks were in the exact situations of the normal blackhead lesions, and in some cases had been restricted by the line of demarcation between the normal and necrotic tissue. They were on the liver and the cæcal wall, and in some cases on the gizzard wall, and consisted of masses of cells of the lymphoid series collected together and closely resembling tumours.

The fact that the cell masses were present in these situations suggests the following hypothesis: The heterakis worms present in the alimentary canal and particularly the cæca damage the intestinal wall and allow the protozoan *Histomonas meleagridis* to reach the liver via the blood stream, and produce the liver lesions. In the cæca and other parts of the alimentary canal also ulcers are formed due to the presence of the histomonads within the connective tissue. Great demands are made upon the actively functioning tissue which produce lymphocytes, and owing to the repeated irritation due to the heavy parasitic infestation these cells are liberated into the blood stream in an immature form and assume the characteristics of tumour cells.

These cases surely support the opinion that in some cases the primary condition is irritation due to parasites, in these particular cases heterakids and histomonads, and lymphomatosis is the result of the demands made upon the blood-forming organs for more lymphocytes than the actively growing bird can normally produce. Possibly the severity of the parasitic invasion is the primary determining factor, whether these immature lymphoid cells are or are not conveyed to the site of the lesions; after which they may spread among young birds without the necessity of a particularly severe infestation.

My experience has been that lymphomatosis uncomplicated by another condition is of very rare occurrence in poultry. It seems reasonable to suppose that the high mortality associated with lymphomatosis is the result of the combination of more than one factor, and its occurrence and the degree of seriousness are very much dependent upon the methods of management and environmental conditions under which the birds are kept.

Hereditary influences can hardly be held to be responsible in any way for lymphomatosis in turkeys; when it occurs it is more likely the result of intensive methods of poultry husbandry supplying the necessary combination of factors which bring about the disease.

The photographs show the distribution of the lesions: Fig. I shows the lesions on the cæcal wall to the left of the liver; Fig. II shows extensive lesions on the cæcal wall. In this photograph the cæcum is arranged at the base of the liver, and large lymphomata are present in the liver.

FIG. 1

FIG. 2

FIG. 1 shows the lesions on the cæcal wall to the left of the liver. FIG. 2 shows extensive lesions on the cæcal wall. In this photograph the cæcum is arranged at the base of the liver, and large lymphomata are present in the liver.

[*To face page* 90

HÆMOCYTOBLASTOSIS*

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Literature

A SURVEY of the literature on fowl paralysis in this country fails to show one paper dealing with hæmocytoblastosis, and therefore as far as can be ascertained this is the first attempt to confirm Emmel's work in the British Isles.

Johnson (1934) was the first to describe it in connection with the development of fowl paralysis or leukæmia, and the term was then used to designate the presence of hæmocytoblasts in the peripheral circulation and the proliferation of those cells in the bone marrow. However, Emmel in his publication dealing with the ætiology of Fowl Paralysis, Leukæmia and Allied Conditions in Animals, *Technical Bulletin* 306, University of Florida (1936), states: "In this study hæmocytoblastosis is regarded as being designated by the presence of a variable number of immature blood cells of any type in the peripheral circulation." Emmel considers that this viewpoint does not differ materially from that of Johnson, but adds: "In addition to immature forms of blood cells of any type, a variable number of degenerative forms of mature and immature blood cells are also found in the peripheral circulation. . . . Hæmocytoblastosis is also characterised by a variable increase in the total number of white cells . . . the erythrocyte count in active hæmocytoblastosis is usually at least slightly under normal."

Emmel states that the condition in chickens may be caused by a number of agents, and that the stimulus necessary to arouse the hæmotopoietic system to action is not great. He considers it to be a variable process, and has shown that it occurs after the intravenous injection of *Salmonella* bacteria, and following the oral exposure of birds to the same causal micro-organism during chronic intestinal parasitism. Dead bacteria (*S. ærtrycke*) are also capable of inducing hæmocytoblastosis when injected intravenously or intraperitoneally.

Perhaps the most important statement in the whole of Emmel's paper is that referable to the actual hæmocytoblast cell itself, in which he says (p. 10): "The *immature lymphocyte* is in all probability an undifferentiated cell and one to which Johnson has referred to as the hæmocytoblast." In an earlier *Bulletin*, No. 284, of the same University, August, 1935, he describes the immature lymphocyte as a cell "closely resembling the small lymphocyte, sometimes slightly larger, but with cytoplasm not quite so basiphilic, and the nucleus is more vesicular, staining less intensively." Unfortunately, this is

*The work reported in this paper formed part of a thesis submitted for the degree of Ph.D. of Edinburgh University.

the only description of the cell given by Emmel, and although he gives diagrams of most of the blood cells seen in hæmocytoblastosis he fails to illustrate the particular cell itself.

As a fact, Emmel seems to have classified the blood cells to his own liking rather than conforming to recognised methods, and indeed states (p. 25, *Bulletin* 284): "Most of the numerous investigators who have made differential counts of blood smears from paralysed or leukæmic birds have used a different classification of blood cells. The classification used in this study is again different. While the author does not claim the classification used herein to be the best, it is simple and appears to give the best understanding of the blood picture in both natural and experimentally induced cases of fowl paralysis and leukæmia."

It would probably be more true to state that Emmel's method has caused the greatest confusion possible, because of its unnecessary divergence from accepted principles, and that, simple though it may be to the author himself, to others who have to rely on the description and diagrams given it is sufficiently confusing to cause a serious misunderstanding of the true blood picture.

In an endeavour to give a name to every cell appearing in the circulation, Emmel has failed to realise that the blood picture is only a mirror of the changes occurring elsewhere (notably in the marrow), and that the disease process is not really in the blood-stream but in the formative organs. This may appear harsh criticism, but the following notes will show clearly Emmel's confused ideas.

Bulletin 284, p. 25: "Leukæmia.—The author's conception of the blood picture in leukæmia is that the fundamental change is one in which there is a degeneration of blood cells with a subsequent effort on the part of the bird to replace these degenerative cells with normal cells. The need for these normal cells is so urgent that immature erythrocytes, leucocytes or both are found in the circulating blood."

Emmel clearly considers the fundamental factor to operate on the cells in the blood rather than on the marrow.

P. 26: "*Basophilic Polymorphonuclear Leucocyte* (mast cell).—This cell contains the characteristic polymorphonuclear nucleus, the cytoplasm containing closely packed basophilic granules."

Since the mast cell nucleus is never polymorphic but always monolobular, and further that it is characterised by closely packed granules which overlie the nucleus, it is improper to refer to the cell as having a typical polymorphonuclear nucleus.

P. 27: "*Mature lymphocyte.*—No differentiation is made between the large and small lymphocyte. In some instances the large lymphocytes greatly outnumber the small lymphocytes. As yet no particular significance has been attached to any change in the numerical relation of these cells."

It can be seen that no appreciation is given to the genetic relationship between these two types of lymphocytes. Further, from a study of Emmel's drawings, although no measurements or scale are given, it is clear that what he describes as the large lymphocyte is really only one of medium size. Then when actually describing a large lymphocyte he refers to this as the monocyte, and finally he introduces the "*myelocyte*"—"usually larger than the monocyte . . . the general structure of the cell closely resembles that of the monocyte."

Perhaps the greatest error in Emmel's description of the blood cells is that of the *Vacuolar Lymphocyte*: "The vacuolar lymphocyte is apparently a cell which McGowan (1926) has described as the fusiform cell. They often appear in groups."

From the complete histological description which follows, together with the diagram and the reference to McGowan's fusiform cell, it is absolutely clear that Emmel is really describing the *thrombocyte*.

This has no relationship to the blood lymphocyte, and elsewhere in the paper Emmel writes: "Thrombocytes have received no consideration in this paper."

From his description of the *Immature Lymphocyte* Emmel is describing an atypical, primary micro-erythroblast, which in the study in question is understood to represent the original stem cell or hæmocytoblast.

The fact that Emmel has labelled the cells incorrectly and introduced terms unnecessarily does not mean that there is no such condition as hæmocytoblastosis. Indeed, it is only too clear that the blood of chickens, under the stress of certain disease conditions, does show abnormalities apparently identical with those which Emmel has grouped collectively as hæmocytoblastosis. However, his original description is not a happy one, for it is very indefinite, namely, "a variable number of immature blood cells of any type." Since no indication is given as to the extent of the immaturity, nor yet how many types require to be involved before the picture can be termed hæmocytoblastosis, the definition is clearly too vague.

Original Observations on Hæmocytoblastosis of Poultry

Hæmatology.—The series of cases examined show in the blood three important features: (1) the presence of cells normally found in the bone marrow and never seen in the peripheral circulation of healthy birds; (2) degenerations involving all types of blood cells; and (3) a total white cell count which, though frequently higher than normal, is not necessarily one of leucocytosis.

With the exception of this last statement, it will be seen that the general blood picture is similar to that described by Emmel. There is no suggestion, however, that the peripheral circulation contains numbers of "hæmocytoblasts" or that there is proliferation of such cells in the marrow, as described by Johnson.

Although hæmocytoblasts as such do not circulate, since the condition is one in which immature cells of all series appear peripherally, the term hæmocytoblastosis does not appear to be wrong if used only in its descriptive sense.

Although the specific feature of hæmocytoblastosis is the presence of immature cells circulating in the blood, the picture obtained is never that of true leukæmia, because neither myeloblasts nor lymphoblasts appear. The earliest granulocytes seen are premyelocytes—which in the fowl have a characteristic appearance—whilst on the lymphatic side of the picture there is nothing more immature than a large lymphocyte. However, as far as the red cells are concerned, erythroblasts commonly appear — often even the primary erythroblasts characterised by their deep blue cytoplasm. One may, therefore, consider the condition to be a mild form of erythroleucosis involving the whole reticulo-endothelial system, for on occasions pro-monocytes are also seen.

At this point it is advisable to consider the terminology used by Emmel in his various papers on the subject in order to show the corrections advocated :

Emmel's terms	*Probably represents*
Hæmocytoblast	Atypical micro-erythroblast
Basophilic erythroblast	Primary erythroblast
Rod-bearing eosinophilic polymorphonuclear	Neutrophile (" polymorph ") granulocyte
Granule-bearing eosinophilic polymorphonuclear	Eosinophile granulocyte
Polychrome erythrocyte	Immature (polychromasic) erythrocyte
Basophilic polymorphonuclear leucocyte	Basiphile granulocyte
Small lymphocyte	Small lymphocyte
Large lymphocyte	Medium-sized lymphocyte
Monocyte	Large lymphocyte
Monocyte showing granules ...	Eosinophile metamyelocyte
Myelocyte	Monocyte (?)
Vacuolar lymphocyte (fusiform cell)	Thrombocyte
Immature lymphocyte	Atypical micro-erythroblast
Mature lymphocyte	Large or small lymphocyte
Premyelocyte	Premyelocyte
" Budding "	Cytoplasmic aggregations—notably in the lymphocytes

The degenerations encountered involve all types of circulating cells and consist of : vacuolation, lysis and clumping or budding of the cytoplasm; cell distortion generally—sometimes leading to rupture; nuclear pyknosis; basiphilia of the cytoplasm of the lymphocyte-monocyte series; atypical granules in

the leucocytes; cell inclusions; formation of erythroplastids and distortions of the periphery of the erythrocyte nucleus leading to "notches."

An additional feature of the blood picture concerns the actual size of the various cells, for a marked feature of the present study has been their great variation and lack of uniformity. Anisocytosis, as is well known, is a term usually restricted to excessive variations in the size of red cells, but its use in a wider sense might be advocated here, because there are just as great differences in size with nearly all the circulating cells—notably the granulocytes.

Poikilocytosis is not a common feature in diseases of avian blood, but erythrocytes more easily distorted than the majority and with pointed ends are to be seen in some cases of hæmocytoblastosis.

The condition may, therefore, be visualised as one in which numbers of immature cells—normally retained in the marrow for maturation purposes—pass into the blood-stream hurriedly. Finding their new environment unsuitable for normal development purposes, they quickly show degenerative changes. It does not seem clear whether they are formed hurriedly and so, being of imperfect structure, they degenerate rapidly; or whether their blood-stream environment—the plasma—should be considered unhealthy, resulting in the collapse of normal cells. The anisocytosis mentioned above is perhaps a point to be considered in favour of the theory that there is undue stimulation of the marrow. Such cells would almost certainly appear if there was amitotic division of their immediate precursors—hence the finding of numbers of cells of all series of unequal size in the general circulation.

Perhaps the most difficult task is to decide when a given blood picture should be called one of hæmocytoblastosis. How far must it differ from the normal? What of the finding of one or two premyelocytes, with perhaps a number of vacuolated lymphocytes? Fortunately, the answer is seldom difficult, for there is usually such a variety of well-marked changes, few cases presenting themselves which leave any doubt as to the diagnosis of hæmocytoblastosis.

There are over twenty features which may be noted in cases of hæmocytoblastosis; therefore, as a rough guide, if any ten or more of these are present the diagnosis should be positive. There should be no doubt at all, of course, if both erythroblasts and premyelocytes are present in the same film.

An examination of large numbers of poultry bloods will show that certain of the features of the blood in hæmocytoblastosis do occur normally—though to a very limited extent. Thus, erythrocyte degeneration, pyknosis and some degrees of anisocytosis are apparently physiological, and although the stimulus for reticulosis (polychromasia) apparently need only be slight, the finding of an erythroblast of any type in the blood-stream is extremely rare. In making an *estimate* of the total leucocyte count (by ascertaining the average number

of white cells per field), if notes are also made of the number of degenerate cells (or other special features seen), then, after examining from ten to fifty such microscopic fields—according to the case—a clear impression will be gained of the general blood picture.

The following are the chief points found in a hæmocytoblastosis blood picture, contrasted with that from a healthy bird:

Granulocytes	*Normal*	*Hæmocytoblastosis*
Toxic granules	0	++
Ruptured cells	0—+	+++
Immature type granules	0—+	+++
Deficiency of granules	0—+	+++
"Anisocytosis"	0—+	+++
Immature types (pregranular types; myelocytes; etc.)	0—+	++++

Lymphocytes	*Normal*	*Hæmocytoblastosis*
Vacuoles in large variety	0	++
Vacuoles in medium variety	0—++	+++
Vacuoles in small variety	0—+	+++
Very small varieties	0—+	+++
Azur granules or masses	0—+	++
Türk cells	0—+	+++
"Budding" of cytoplasm	0—+	++++

Monocytes	*Normal*	*Hæmocytoblastosis*
Vacuolated	0	0—+
Türk-type	0	0—+
"Anisocytosis"	0	++

Erythrocytes	*Normal*	*Hæmocytoblastosis*
Pyknosis	0—++	++++
"Notches"	0—+	+++
"Anisocytosis"	0—++	+++
Poikilocytosis	0—+	+++
"Plastid" formation	0	+++
Regeneration types	0—+	++++
Degeneration types	+	+++
Erythroblasts (polychromasic)	0—+	++++
Primary erythroblasts (basic)	0	++

Thrombocytes	*Normal*	*Hæmocytoblastosis*
"Anisocytosis"	0—+	+++
"Poikilocytosis"	0—+	++
Condensation of the cytoplasm	0—+	+++
Pyknosis	0—++	+++

Hæmocytoblastosis and Coccidiosis

A comparison of the hæmocytoblastosis picture in ten chicks suffering from typical cæcal coccidiosis contrasted with an equal number of cases involving the small intestines in pullets shows several interesting features.

In the duodenal coccidiosis cases there is a leucocytosis—averaging about 35,800 p.cm.—whereas in the cæcal type there is a definite leucopenia with only about half this number present, i.e., 16,450 cells/c.mm. This is contrary to the experience of Emmel, who found leucocytosis an invariable accompaniment of hæmocytoblastosis. Since the number of leucocytes was often quite high, it must be assumed that there was operating in his cases a much more potent stimulus to the marrow. There is little doubt this would be due mainly to the nature of his experiments, i.e., that of inducing a *Salmonella* septi-

cæmia; whereas contrasted with that the preceding cases were natural instances of early hæmocytoblastosis.

In both types of coccidiosis, the myeloid response is best seen where the red cells are concerned for erythroblasts of all types occur, together with other signs of active regeneration. At the same time the destruction of mature erythrocytes in the blood-stream often appears increased, notably in cæcal coccidiosis, where there are often large numbers of nuclear remnants to be found.

Premyelocytes occur in more cases of cæcal than duodenal coccidiosis, as do the pre-granular basiphilie myelocytes, and there is also considerable variation in the size of the monocytes. In duodenal coccidiosis, many cases show a characteristic degeneration of the polymorphs, in which the rod-shaped granules appear as "clubs"—and, in some of the eosinophiles, the granules lose their natural round contour, to become semi-neutrophilic in type.

In this study, the lymphocytes show little of comparative value for Türk cells, vacuolations and "budding" are common to both types of coccidiosis. The cell described by Emmel as an immature lymphocyte—actually a small erythroblast with basic cytoplasm—occurs in about 45 per cent. of all cases.

No.	*Degree of cæcal coccidiosis*	*Age (weeks)*	*Notes*	*Degree of hæmocytoblastosis*
1	+++	7	pus +	+
2	+++	12	acute	+
3	+	6	post-cocc.	++
4	+++	4	acute	++
5	+++	6	chronic	+++
6	++	4	acute	+++
7	++	10	chronic	+++
8	++	4	acute	+++
9	+	12	post-cocc	+++
10	None	6	—	++++
11	+++	4	—	++++
12	++++	4½	—	++++
13	++++	4	—	++++
14	++	4	dying	++++
15	+	6	killed by cannibalism	++++
16	++			

No.	*Degree of duodenal coccidiosis*	*Age (weeks)*	*Notes*	*Degree of hæmocytoblastosis*
16	++	20	ascaridia ++	+
17	++	4½	cæcal ++	++++
18	+	6	—	+++
19	+	12	davainea ++ capillaria + cæcal +	+++
20	++	10	—	+++
21	+++	16	cæcal +++	+++
22	++	18	advanced nephritis	+++
23	++++	12	davainea +	+++
24	++	11	heterakis + cæcal ++++	++++
25	+++	10	cæcal ++	++++
26	+++	10	—	++++

It will be seen from the foregoing table that the degree of hæmocytoblastosis does not appear to bear a direct relationship to the disease conditions, but this is somewhat to be expected because it is probable that hæmocytoblastosis is an after-effect of coccidiosis, rather than an accompaniment of the disease. The condition should therefore be visualised as a sequel to coccidiosis and not a direct symptom of the disease. The present study does not disclose whether uncomplicated coccidiosis alone can result in blood changes characteristic of hæmocytoblastosis, or whether, following an infestation by protozoa, bacteria require to gain access to the tissues—there to multiply, produce toxins and so stimulate the bone marrow. Emmel's theory is to the effect that the degree of hæmocytoblastosis depends on the number of causal micro-organisms entering the blood-stream, their rate of entry, period over which they enter, and potency. He considers that recovery from hæmocytoblastosis is a slow process requiring as long as from three to six months, but he gives no figures for the "incubation period," although the growth curve of artificially induced cases in three-week-old chicks shows the process to be well established after about ten to twenty days.

Case 9 in the same table is of special interest, because the post-mortem examination revealed no coccidiosis, although the blood picture was one of advanced hæmocytoblastosis. This bird was taken from a flock where cæcal coccidiosis had been rife, and from the clinical standpoint it appeared quite healthy, although perhaps slightly undersized. It is not improbable that the bird did suffer from a mild attack of coccidiosis, afterwards to recover. The development of hæmocytoblastosis may have occurred as a direct result of the parasites, or only following the establishment of some bacterial focus. However, what is abundantly clear from the present study is that the effects of coccidiosis are not limited to the damage directly associated with the parasites as seen at post-mortem examination. Therefore, it is impossible to assess accurately the state of health of chicken following an outbreak of coccidiosis by noting their general appearance and weight without considering at the same time an examination of the blood cells.

Hæmocytoblastosis and Fowl Paralysis

Emmel believes that hæmocytoblastosis is an essential factor in the development of fowl paralysis, although in itself he does not consider it will necessarily lead to paralysis—other factors require to be correlated with it.

Twenty-four cases of fowl paralysis have been studied and the degree of hæmocytoblastosis noted. In fourteen instances there were no blood changes considered sufficiently important to be classed as evidence of hæmocytoblastosis, and in the remaining ten only two showed advanced features, thus:

Evidence as to hæmocytoblastosis		*24 fowl paralysis cases*
Advanced (+++)	=	2
Moderate (++)	=	3
Slight (+)	=	5
None (—)	=	14

No relationship was noted between the degree of hæmocytoblastosis and the extent or type of the fowl paralysis lesions, nor were the blood changes directly associated with the number of parasites present.

Contrasted with the previously mentioned cases of hæmocytoblastosis (associated with coccidiosis), the stimulation of the marrow in fowl paralysis appears to be far less severe, for erythroblasts and premyelocytes were not numerous. Degenerative changes were marked, both in the erythrocytes and lymphocytes, particularly noteworthy being the large numbers of erythrocytes with indentations or pyknosis of the nucleus. A number of others were without nuclei — erythroplastids — and in every case nuclear remnants were prominent. Lymphocytes with "budded" and vacuolated cytoplasm were common, as were Türk cells. The average white cell count was 37,500 cells/c.m.—a leucocytosis much the same as that noted in the cases of duodenal coccidiosis.

The main features are shown on the accompanying tables.

Case Reference	314	225	333	280	128
Primary erythroblasts ...	—	—	—	+	+
Polychromasic erythroblasts	—	—	—	++	+
Regenerative r.b.cs.	++	—	—	+++	+++
Degenerative r.b.cs.	+	+	+	+	+
Nuclear remnants	+	+	+	+	+
"Notches"	+++	—	+	+	—
"Plastids"	+	—	++	+	+
Pyknotic r.b.c.	++	—	++	+	—
Anisocytosis	++	++	—	++++	++
Thrombocytes	+++	—	—	—	++
"Budding"	+++	+	+++	+++	++
Vacuolation	++	—	++	++	—
Türk cells	—	—	+	—	+
Premyelocytes	++	—	+	—	—
Toxic granules	—	++	—	—	—
Basiphiles	—	—	—	+	—
Hæmocytoblastosis	+++	+	++	+++	++
Leucocytes (000's)	37	68	34	57	10.5
Fowl paralysis	Early ++	Tumours ++++	F.P. ++	F.P. ++	Very early +
Parasites	++	—	++	—	++

Case reference	201	298	294	219	299
Primary erythroblasts ...	—	—	—	—	—
Polychromasic erythroblasts	—	—	—	+	—
Regenerative r.b.cs.	+	—	—	++	+
Degenerative r.b.cs.	++	+	+	+++	+
Nuclear remnants	++	+	++	+++	+
"Notches"	+	—	++	—	—
"Plastids"	—	—	—	++	—
Pyknotic r.b.c.	—	+	—	+	—
Anisocytosis	—	+	—	—	—
Thrombocytes	—	++	—	—	—
"Budding"	+	+++	++	+	—
Vacuolation	+	+	+	+	+
Türk cells	—	+	—	+	+
Premyelocytes	—	—	—	—	—
Toxic granules	—	—	—	—	—
Basiphiles	—	+	—	—	—

Case reference	201	298	294	219	299
Hæmocytoblastosis	+	+	+	++	+
Leucocytes (000's)	28	19.5	14	45	44
Fowl paralysis	F.P. +	advanced +++	advanced +++	eye +++	Eye +++ F.P. +
Parasites	—	+	—	++++	++++

Hæmocytoblastosis in Day-old Chicks

Hitherto, hæmocytoblastosis has been regarded as a disease phenomenon, but examinations of day-old chicks show that a similar process is operating at birth. The blood counts of twenty day-old chicks—some healthy and others too weak to break out of their shell—show changes identical with those recognised as hæmocytoblastosis. As a fact, the response of the marrow is, however, greater where the red cells are concerned, for large numbers of primary erythroblasts are found in the circulation. There is great variation in the size of these, and in addition some of the lymphocytes show vacuoles or Türk-type cytoplasm and not a few of the basiphiles are of the early type. A few premyelocytes can also be found, so that a consideration of all these features of the blood shows clearly a picture very little removed from that of hæmocytoblastosis. However, the total white cell count is greatly reduced, so that a marked leucopenia exists.

The accompanying table shows the majority of the blood changes encountered in the physiological hæmocytoblastosis of seven typical day-old chicks.

HÆMOCYTOBLASTOSIS—DAY-OLD CHICKS

TABLE XXII

Reference	235	234	246	232	245	233	244
Primary erythroblasts ...	+	+	+	+++	+++	+	+
Polychromasic erythroblasts ...	++	++	+++	++++	+	—	++
Regenerative r.b.c.	+++	++++	++++	++++	++	+++	+++
Degenerative r.b.c.	++++	++	+	+++	++	+	++
Nuclear remnants ...	++++	++	+	++	++	+	+
"Notches" ...	—	—	—	—	—	—	—
"Plastids" ...	+	+++	++++	+	+++	++	++++
Pyknotic r.b.c. ...	—	—	+	—	++	+	—
Anisocytosis ...	—	+	++	—	—	—	—
Platelets ...	—	—	—	—	—	—	—
Budding ...	—	—	—	—	—	—	—
Vacuolations ...	—	—	—	—	—	—	—
Türk cells ...	++	+	+++	+	—	—	+
Premyelocytes ..	—	+	—	—	—	—	—
Toxic granules	++	—	—	++	—	—	+
Basiphiles ...	—	—	—	—	+	—	—
W.b.cs.	9.2	9.0	9.5	8.5	5.0	9.5	6.8

Other Examples of Hæmocytoblastosis in Poultry

Following injections of a potent *liver extract* into pullets, there occurs a temporary stimulation of the bone marrow. This is reflected in the blood

picture by the appearance of a small number of erythroblasts—both primary and polychromasic—and there are also other regenerative signs. Immature leucocytes or degenerative forms may also be found, therefore this response may be considered to represent an *early* type of hæmocytoblastosis. Similarly, young chicks aged 2-4 days suffering from the effects of severe *chilling* show blood changes typical of hæmocytoblastosis.

An 18-day-old chick, a survivor from an outbreak of *bacillary white diarrhœa*, has also been found to show in the blood typical signs of hæmocytoblastosis. Erythroblasts and erythroplastids were present, whilst some other red cells were immature or with pyknotic nuclei. Monocytes, lymphocytes and a few erythocytes were vacuolated, as was an occasional neutrophile.

Here, then, was an example of hæmocytoblastosis showing early red cells and degenerate red and white cells, but no early leucocytes.

As a contrast, a bird suffering from *fowl pox* showed numerous premyelocytes and degenerative leucocytes, but no anæmic features.

Finally, typical changes representative of advanced hæmocytoblastosis have been noted in a fowl suffering from *leukæmia* (myeloid) with dropsy, and also in *blackhead* in turkeys.

Summary of Observations on Hæmocytoblastosis in Poultry

From a full consideration of the foregoing statements, it is possible to summarise the available knowledge as follows:—

So-called "hæmocytoblastosis" is an example of myeloid stimulation in poultry. It is characterised (in its advanced typical form) by the finding of immature or degenerate red and white cells in the peripheral blood circulation. It is non-specific, occurring in chicken of all ages, breeds and in both sexes; and, although commonly encountered in the domestic hen, it has also been observed in the turkey.

Contrary to the experience of Emmel, hæmocytoblastosis is not always associated with an increase in the total number of white cells, nor is it necessarily of pathogenic origin, for identical changes are seen in the blood of healthy day-old chicks. Apart from this physiological occurrence of hæmocytoblastosis, it has been observed in chilled chicks, following injections of liver extract, and in diseases of virus, bacterial and parasitic origins, and although erythroblasts are nearly always seen in typical cases of hæmocytoblastosis, the leucocyte response is far less intense, for neither myeloblasts nor lymphoblasts appear in the circulation, but premyelocytes are always found when the process is advanced.

All stages between the typical hæmocytoblastosis picture of Emmel and normal blood counts have been recorded, and therefore hæmocytoblastosis is considered to represent a type of myeloid response in poultry, and not a disease *per se*.

It has not been proved in the present series of cases that hæmocytoblastosis

is an essential factor in the development of fowl paralysis, for it does not appear as a constant finding in blood examinations of birds suffering from this disease, but it would rather appear to represent a post-coccidial manifestation. Since coccidiosis, in some form or degree, precedes fowl paralysis in large numbers of cases, the association between hæmocytoblastosis and fowl paralysis is probably more directly connected with the damage of the coccidial parasites than with any specific neurotropic virus sometimes postulated as the specific cause of neuro-lymphomatosis-gallinarum.

In view of the greater response of that portion of the bone marrow associated with erythrocytopoiesis, it is probably more correct to refer to the condition, in its typical form, as erythroblastosis.

The percentage distribution of the main features characteristic of hæmocytoblastosis in new-born chicks and in cases of coccidiosis and fowl paralysis is shown on the accompanying table.

HÆMOCYTOBLASTOSIS IN THE DOMESTIC HEN

	Normal day-old chicks	*Cæcal coccidiosis*	*Duodenal coccidiosis*	*Fowl paralysis*
	%	%	%	%
Primary erythroblasts	100	60	70	20
Polychromasic erythroblasts	100	70	80	30
Polychromasic erythrocytes	100	70	80	60
Nuclear remnants	100	90	100	100
Pyknosis	40	60	70	50
Anisocytosis	40	40	50	50
Erythroplastids	100	80	60	50
"Notches"	0	50	40	50
Türk cells	70	70	60	50
Vacuolated lymphocytes	0	40	50	80
Hæmocytoblasts	40	30	60	20
Leucocytosis	0	70	20	60

ANÆSTHESIA IN POULTRY

By ERNEST GRAY, M.R.C.V.S., F.R.M.S.

Lecturer in Veterinary Hygiene, East Anglian Institute of Agriculture, Chelmsford, Essex

DURING some recent researches into paralysis of fowls it was found necessary to examine the spinal fluid and to resect nerve trunks such as the brachial and sciatic.

For this purpose narcotics were necessary, as it was essential these examinations should be carried out during life.

Little has been written as to the tolerance of poultry to the common narcotics and anæsthetics, and it was found that their tolerance is quite different from that of the common four-footed animals met with in veterinary practice.

Two generalised narcotics were first used, one a barbituric acid preparation not yet put on the market.

The dose for dogs and cats of this drug was 70 milligrams per kilogram body weight. Administration of this quantity intraperitoneally into fowls produced no results. The minimum lethal dose, 100-110 mgs. per kg. body weight was then given intraperitoneally. Again no results were visible. Finally twice the lethal dose was given. 200 mgs. kg. weight were given hypodermically and intraperitoneally, but no effects were noted until after 13½ minutes following administration. The bird then stretched itself, flapped its wings, and chattered its beak. At 14 minutes the body began to droop, but no signs of narcosis appeared till 34 minutes after the injection when it suddenly went off its legs. At 44½ minutes it was asleep on its side when breathing was normal and reflexes were well established. At one hour 40 minutes from the commencement of the experiment breathing became shallow, but reflexes were still well established. At the end of the second hour breathing again became deeper, and an uneventful recovery was made within three hours from the primary injection.

The preparation was then abandoned, and nembutal tried. The dose was estimated from that suggested by weight, one grain per five lbs. body weight. The first bird weighed between two and a half and three pounds, and half a grain was accordingly given in solution intraperitoneally.

After five minutes the bird was sleepy, in seven the wings were relaxing, in nine minutes the head was relaxed and laid on the ground. After 17 minutes there was generalised narcosis but the bird remained extremely sensitive to noise and the reflexes were not abolished. The bird remained drowsy for eight hours.

One grain was then given to a Black Leghorn weighing about four pounds. The eyes began closing in 3½ minutes, and there was "air hunger" shown by the bird raising the beak and gasping. In six minutes it settled down, but although drowsy it did not become so narcotised as the first bird, and had quiescent followed by waking periods, when it would raise the head and look round it. Recovery occurred within five hours.

Nembutal was used in several instances afterwards, but it was never found to produce a degree of narcosis sufficient to carry out surgical operations. The birds went under, but recovered in a manner similar to the two first cases, and even when ether vapour was also administered the birds were only lightly anæsthetised.

A ten per cent. solution of chloral hydrate was given orally to one bird, and produced complete cessation of movement and extreme drowsiness within seven minutes. The reflexes, however, never became abolished, and the bird was extremely sensitive to sound. It remained in a deep sleep for over three hours, however.

Chloroform was found to be the most effective anæsthetic. Some

workers have stated that birds are almost insensitive to this drug, but the writer found that concentrated chloroform vapour killed a bird of six pounds within two minutes.

A pad of cotton wool soaked in chloroform was given to a second bird in a rubber surgical glove, care being taken that the bird's head was not in contact with the wool. Its eyes could be seen through the rubber, and it fell asleep in two minutes twelve seconds. At four minutes there was relaxation, and in five and a half minutes cessation of breathing. The "mask" was then removed, when dyspnœa appeared. The bird again began breathing normally, and showed some signs of recovery.

Subsequently chloroform was given mixed with air by means of a cotton wool pad soaked in chloroform placed in a wide-mouthed glass jar.

The bird's reactions could be watched through the glass and it was found that by careful administration, anæsthesia could be provoked within four minutes and a bird kept under perfect deep anæsthesia for from 30 to 40 minutes. There is a definite period of excitement, occurring about three minutes after administration, but it appears to vary considerably in different birds.

Anæsthesia after 40 minutes appears to be dangerous, many birds then recovering, or passing insensibly into a cessation of breathing which is very difficult to detect. It is possible that the tolerance of chloroform vapour is dependent upon the fat content of the liver, and as in many birds this organ is diseased there may be an associated intolerance to prolonged dosage.

The respiratory movements of poultry were found to be no indication of their degree of anæsthesia. Birds with shallow intermittent respirations remained alive, whilst others in which the breathing was deep and regular died abruptly. This again may be a feature of their physiology, the lungs of birds being embedded into the ribs and the most powerful respiratory movement being expiration, not inspiration.

About sixty birds have been exposed to one or other of the above narcotics, the greater proportion to chloroform. From the practical point of view chloroform is the only drug which appears to give consistent deep anæsthesia.

LYMPHOMATOSIS (FOWL PARALYSIS) IN THE PHEASANT

By S. T. HARRISS, M.R.C.V.S., B.Sc., D.V.S.M.
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THE final proof of the theory of an infectious agent as causative of lymphomatosis in fowls must depend upon the critical evidence supplied by transmission experiments. Such experiments, in the past, have proved in-

conclusive because the disease, to a less extent and in a variable degree of incidence and severity, has commonly appeared in the uninoculated controls. This almost constant result may tentatively be interpreted in several ways. It might be due to the disease being latent in the birds of both inoculated and control groups or it might be due to accidental infection during the course of the experiment. Any increased incidence in an inoculated group might be attributed to the possibility of inoculation lowering the resistance of the bird to a latent, non-infectious disease.

It seemed reasonable to suppose that the use, for experimental purposes, of a bird in which lymphomatosis does not commonly occur, might reveal the possibility of producing the disease by inoculation, while uninoculated controls remained free. The success of such a venture would enable a further study of the conditions under which inoculation succeeded. It would make it possible, for example, to differentiate between the efficacy of the implantation of neoplastic cells and the inoculation of a cell-free filtrate.

The promise of this line of investigation suggested the use of the pheasant and, with these ends in view, 62 pheasants were hatched and reared at the Rowett Institute in the summer of 1938. Forty-two of these were inoculated by several routes while the remainder were left uninoculated as controls. The experiment is not yet completed—the present communication dealing merely with an undoubted case of lymphomatosis which has occurred in one of the inoculated pheasants, proving, therefore, that the disease may occur in this species.

Subject

A hen pheasant of unknown strain. Age: 134 days.

History

This bird was hatched in an incubator and reared in a hover, afterwards being kept and maintained with others in a wire netting enclosure. At no time had there been direct contact between any of these birds with farmyard poultry, though affected fowls were at no great distance away. This bird formed one of a group of 62 pheasants intended for experimental work on lymphomatosis.

At 14 days old it was inoculated intraperitoneally with 0.1 ml. of a 10 per cent. nerve suspension derived from two clinically paralysed fowls—a White Wyandotte pullet and a White Leghorn cockerel.

For six days prior to its death the bird was noticed to be ill and was kept under observation. It was finally killed by asphyxiation with coal gas.

Symptoms

Inco-ordination of gait; trailing of the left wing and drooping of the right; dyspnœa, characterised by gasping respirations through the open beak; head held on one side, with right side downwards; complete loss of appetite;

considerable thirst; sneezing and coughing. There was no tendency to use its wings for flight though the wings had never been clipped or bound. Throughout the period of observation the bird remained alert and conscious of the presence of danger.

Post-mortem Examination

The tip of the tongue was noticed to be necrotic. Macroscopic examination of the organs and carcase, which was well nourished, revealed nothing of pathological significance. There appeared to be a slight suggestion of translucency in both vagi but there was little suggestion of enlargement in these or other nerves. The contents and scrapings of cæca and duodenum were both negative for coccidia. The ovaries were inactive.

Histological Examination

Marked lymphocytic infiltration was found in both vagi and in the left brachial plexus. Considerable perivascular infiltration of a lymphoid character occurred in the liver and kidneys. (See accompanying photo-micrographs).

Conclusion

A case of lymphomatosis following inoculation is recorded in a pheasant. It is not as yet claimed that the occurrence of the disease resulted from the inoculation.

The author desires to express his indebtedness to Dr. A. H. H. Fraser for helpful advice and criticism, to Mr. J. W. Johnston for his care and attention in the successful artificial rearing of this group of birds, to Dr. J. T. Irving for his skill and care in the production of the photo-micrographs and to Mr. John C. Grant of Marischal College, Aberdeen, for assistance in procuring material for inoculation.

AVIAN LEUKÆMIA, LEUCOSIS, AND FOWL PARALYSIS

By **ERNEST GRAY, M.R.C.V.S., F.R.M.S.**
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LEUKAEMIA of poultry occurs in isolated instances in a flock, only one or two cases being found per year on a farm. The bird is usually found dead; occasionally there is a short history of wasting, weakness, or disinclination to feed, prior to death.

1.

2.

3.

KEY TO PHOTO-MICROGRAPHS.

FIG. 1.—Left Brachial Plexus x 220.
Infiltration of lymphocytes.

FIG. 2.—Right Brachial Plexus x 220.
No infiltration.

FIG. 3.—Kidney x 220.
Perivascular infiltration.

[*To face page* 106.

XUM

4.

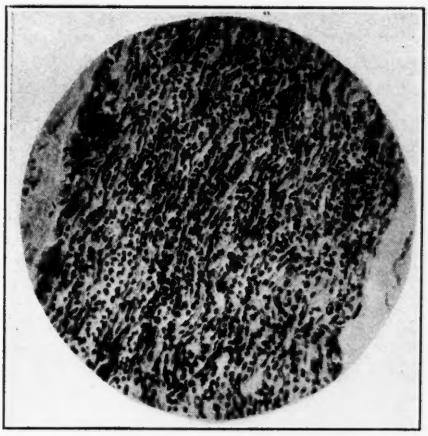

FIG. 4.—Left Vagus x 220. Infiltration.

5.

FIG. 5.—Right Vagus x 220. Infiltration.

[*To face page* 106.

XUM

At post-mortem the liver is found grossly enlarged, firm, and of a universal reddish hue resembling beef-steak; the spleen is also enlarged, and the marrow in the epiphyses of the long bones is dark red in colour, viscid in consistency, and frequently speckled by pale areas of necrosis, or fatty degeneration, giving the marrow as a whole a fanciful appearance to "raspberry jam."

Death is sometimes the result of internal hæmorrhage from rupture of the enlarged liver, or, very occasionally, the spleen.

The cause of the disease is unknown. It may be the result of a virus infection, or more probably is a hyperplasia of the blood forming elements, with resultant hyperplasia of such blood destructive elements as the Malpighian bodies of the splenic pulp.

Leukæmia in mammals is described by some as "cancer of the blood-forming tissues." In man it has been supposed by certain workers to be the result of infection with the bacillus of avian tuberculosis. Attempts to treat cases with extracts of avian tuberculin have, however, not been successful. The form found in poultry must however be carefully distinguished from leukæmia of quadrupeds. Leukæmia of mammals occurs in two forms (a) splenic, medullary, or myolegenous affecting the hæmopoietic systems, its pathological picture resembling leukæmia of fowls; (b) lymphatic leukæmia involving the spleen, bone marrow, and lymph glands. Lymphatic leukæmia is the form thought in human cases to be the result of infection by avian tuberculosis. The blood of patients suffering from lymphatic leukæmia is found to contain an excess of white cells, especially small lymphocytes, and transitional forms. The blood picture merges into true spleno-medullary leukæmia with the appearance of increasing numbers of myelocytes. The latter are an indication that the bone marrow is becoming involved and are present in greatest numbers in medullary leukæmia.

Hodgkins disease is a leukæmia in which the splachnic organs, liver and spleen, and, usually, the bone marrow, are involved, but there is no marked increase in the number of leucocytes. The lymphoid tissue, however, quickly becomes hyperplastic, but associated enlargement of the spleen and bone marrow is slow in appearance. It is doubtful whether true Hodgkins disease occurs in quadrupeds.

Another obscure form of pseudo-leukæmia characterised by a bilateral enlargement of the lymphatic glands is occasionally seen, typically in the dog.

The type of leukæmia present in mammals may be ascertained by blood cell count by the hæmocytometer. In birds, however, this is almost impossible, as the blood picture of fowls varies even in health between very wide limits.

The table overleaf from Burnett's "Clinical Pathology of the Blood"

indicates the range in variations in blood cell counts during health of the horse, dog and fowl, noted by different observers.

	Red Corpuscles millions per cmm.	Leucocytes per cmm.	
Horse	6.5—9.2	7,200—8,800	Bidault
	7.9	5,625	Moore
	7.4	9,500	Hayem
		Average 8,000—Burnett	
Dog	5.9	8,221	Burnett
	7.2	19,000	Dawson
	6.6	10,000	Hayem
		Average 8,000—Burnett	
Fowl	2.4	26,300	Hayem
	3.6	20,081	Moore
	2—3	12—29,000	Warthin
	3.2	19,000	Goodall
	3	30,000	Ellerman & Bang
		Average 18—36,000 Burnett	

Lymphocytes appear to be more numerous in the blood of fowls than in other animals.

	Leucocytes per cmm.	Lymphocytes	Large M.	Polymorphs	Eosins.	Mast Cells.
Ellerman & Bang	30,000	40	23	—	37	—
Goodall	19,000	56	—	37	6	1
Mack	33,777	54.9	12	32.7	2.7	3.3
Burnett	17,921	58	5.5	28.8	3.3	4.3

Little work has been done on the process of repair and scar formation in birds, and it would be interesting to know exactly what role is played by the lymphocytes. It would seem from practical observation that they take an equal part with polymorph leucocytes in all processes of inflammation and (attempted) repair to damaged and/or necrotic tissues; unlike mammals, where polymorphs play the greatest part, usually to the exclusion of lymphocytes.

In leucosis of poultry lymphocytes appear in such enormous numbers that lymphoid tumours occur in the ovary, mesentery, intestinal wall, liver, and other organs. The tumours are white in colour, soft in consistency and vary in size from a millet seed to a Jaffa orange. One or more tumours may be present, or there may be only a diffuse ruguous thickening over the mesentery and peritoneum, frequently associated with dropsy. Histologically the tumours are found to be composed of round cells with practically no basal tissue. The condition has been termed avian lymphatic leukæmia, but there is no enlargement of lymph glands as in mammals.

Leucosis, like leukæmia, may occur in isolated instances, in which case

it is usual to find only one or two tumours at post-mortem. Leucosis may however occur as a generalised condition in a flock, there being symptoms in the birds during life of wasting, unthriftiness, diarrhœa of a brown colour, and infiltration of the anterior chamber of the eye with small round cells that cause an opacity known to farmers as " Pearly Eye."

Leucosis of this generalised nature is alternatively called lymphomatosis, the latter term possibly being more accurate, since there is a preponderance of lymphocytes rather than an equal increase of all white cells of the blood. At post-mortem the typical white " cauliflower " growths are found, the birds most distressed during life being frequently found to have a diffuse leucosis of the mesentery and peritoneum.

In one peculiar form, popularly known as Fowl Paralysis, the lymphocytes invade the great nerve trunks, causing clinical symptoms of paralysis. In such cases it would appear that the spinal fluid also becomes turbid with small round cells, the leucosis of the medullary fluid being found also in birds parasitised but not paralysed. Attempts to detect leucosis during life by blood cell counts are defeated by ignorance of what figure exactly comprises an abnormal white cell percentage.

Leucosis must be carefully distinguished from the condition of contagious sarcomata, which occasionally appears in a flock and runs through it with a mortality varying from six to sixty per cent. In one such case experienced by the author a flock of pullets of six to eight weeks were affected. Birds became unthrifty and wasted, and died on an average in two weeks. No diarrhœa was present, and appetite remained good until the end. At autopsy one or more large white tumours superficially resembling lymphomata were found in the kidney, ovary, and liver. The tumours were, however, creamier in colour, harder, and more discrete in shape. Histological examination revealed them to be typical small-celled sarcoma. The clinical picture was complicated by the presence of miliary tuberculosis which appeared apparently after the primary sarcoma infection. The outbreak flared up, reached a peak, and died down within a period of two to three months, over thirty birds dying out of a flock of ninety. It was assumed that the disease was the result of transmission of the filterable virus of fowl tumours. Two such viruses are known, the Rous and Fujyama, and both produce a sarcomatous lesion. Attempts to ascertain the cell count were made in order to see whether the condition was a form of " Lympho-Sarcomatous Leucosis " but in three birds were found to vary between eighteen and twenty-seven thousand, and were given up. When the survivors were tested for the presence of tuberculosis a few birds gave a reaction to the tuberculin, but at post-mortem were found to be not tubercular, but sarcomatous.

The causative agent of leucosis is unknown. The incidence of the disease however, appears most definitely to be on the increase, as may be seen, for example, by the post-mortem returns from egg laying trials.

In the case of the rous sarcoma the causative agent, a filterable virus, has been the object of considerable study. It has recently been shown that its injection into the blood stream of a healthy fowl does not cause tumour formation, the virus disappearing from the tissues within a fortnight. If, however, the virus is injected into a fowl having some lesion in the muscles, tumours will subsequently appear at the site of injury.

Assuming a virus to be the cause of leucosis; and assuming that in poultry lymphocytes migrate to damaged or necrotic areas, as do the polymorphs of mammals, are the tumours of leucosis the result of vast accumulations of defensive cells on some patch of tissue which has become necrotic through organic cellular degenerations or the effect of such external agents as bacteria and their products?

Abstracts

AN OUTBREAK OF POX IN TURKEYS WITH NOTES ON DIAGNOSIS AND IMMUNISATION

By C. A. BRANDLY and G. L. DUNLAP

THE clinical evidence of the occurrence of pox in this outbreak consisted in the occurrence of lesions on the feet apparently arising from infection with the virus of the injuries caused by toe-punching and in one case a lesion at the commissure of the mouth.

The authors describe four diagnostic methods:

(a) Infectivity tests.—Suspected material was inoculated into the skin of susceptible young chickens. The turkey pox virus in this outbreak only produced mild lesions in these birds and attempts to enhance its virulence for fowls by chick passage were unsuccessful. It was shown by section work, however, that the virus was active in that large numbers of Borrel elementary bodies were demonstrated in the lesions.

(b) Protection tests.—These are valuable if known pox-immune birds are available or if inoculated birds survive, when they can be tested for immunity by a known virus. The authors state that evidence of a partial

immunity may be detected as early as three to four days after exposure (inoculation), but maximum protection is not attained till about three weeks later.

(c) Microscopic methods. — This consists in the demonstration of elementary bodies (Borrel bodies) either in direct smears or sections of the lesions. In direct smears it may be possible to demonstrate large numbers of these bodies, and thus a rapid diagnosis can be made.

(d) Serological methods.—Several methods may be used. A hyperimmune serum can be produced and by its use agglutination tests or actual protection tests can be carried out. Virus propagated in developing eggs or virus "purified" by cerebral passage in chicks may serve as sources of virus for serological work.

In the control of this outbreak, the authors culled out the affected poults and vaccinated the apparently healthy remaining birds (three weeks of age and older) with 0.2 per cent. suspension of pox-infected chorio-allantoic membranes. The vaccination was carried out by making two punctures on the skin of the breast. Good "takes" were observed in ten to fourteen days.

THE COLLECTION OF SPERMATOZOA FROM THE DOMESTIC FOWL AND TURKEY

By W. H. BURROWS and J. P. QUINN

THE method of obtaining semen from the fowl differs from that in other animals because of the former's peculiar genital anatomy. The author had already described a method, and in this article a modification is given in detail. The operation is carried out by two men, though if only one man is available a special holder for the bird can be used. The former method consisted in massaging the soft sides of the abdomen between the gizzard and pelvic bones, which causes ejaculatory response, the semen being caught in a small beaker held under the vent. The modification consists in slightly stimulating the bird as above till the copulatory organ is protruded, and exposing the organ with the left hand fingers so long as muscular contractions persist. The bulbous ducts are squeezed out and the organ is released. The ducts fill up again and the process can be repeated. It is possible to make from two to eight "milkings" at a single collection, and the amount of semen obtainable at the single collection varies from 0.1 to 4 c.c.

Poult. Sc. (1937), XVI, 19.

This method is particularly useful for turkey cocks, the average amount of semen obtained being 2 c.c.

The authors were able to make 124 collections from each of 22 cockerels at intervals of one to three days over a period of about six months.

All cockerels do not respond equally well, and it was noted that males running with females yielded only small amounts of semen, and that males kept separately yielded better than when a group was kept together.

ARTIFICIAL BREEDING OF TURKEYS

By W. H. BURROWS and S. J. MARSDEN

THIS article describes the results of artificial insemination of turkey hens with semen obtained from turkey males by the method of Burrows and Quinn. Mixed samples of semen from 12 turkey males were used (each male contributing from 0.1 to 0.7 c.c.) and 0.05 c.c. was used for each of the 32 hens in the experiment. The hens were divided into four groups and insemination was carried out at periods varying from 1 to 4 weeks—the eggs laid between each insemination being examined for fertility. It was found that of the total of 464 eggs laid during 30-day periods in which only one insemination was made at the beginning of each period, 83.1 per cent. were fertile, and that of the 948 eggs laid in all the other groups (1-3 week periods) the fertility rate was 80 per cent.

A further experiment was carried out on 10 hens running with an old male and whose eggs had given poor results. Semen was collected from the same male and artificial insemination was practised on the same hens. The fertility rate of the eggs increased from 7.5 to 88.4 per cent.

Poult. Sc. (1938), XVII, 408.

THE VALUE OF CHEMICALLY KILLED CULTURES FOR THE CONTROL OF CHOLERA IN DUCKS

By K. F. HILBERT and H. TAX

DISEASE losses on some of the large duck ranches on Long Island may exceed 10,000 in a single year. The methods of rearing ducklings favour the spreading of infectious disease. Much of the loss is from cholera caused

Cornell Vet. (1938), XXVIII, 275.

by Pasteurella aviseptica, and for its control killed culture vaccine has been used. The vaccine is prepared from cultures isolated from the flock on which it is to be used. After 48 hours' growth in plain broth 0.5 per cent. phenol is added and incubation is carried out for a further 24 hours. The doses used are 1 c.c. for ducklings and 2 c.c. for older ducks.

A large experiment was carried out on four ranches, in which 45,411 ducks and ducklings were vaccinated and 5,761 were kept untreated as controls, running with the others. The mortality from duck cholera was 4.1 per cent. in the vaccinated group and 29.4 per cent. among the controls. "It is concluded, therefore, that the procedure is a means of controlling this disease even when birds are kept under the insanitary conditions which prevail on the ordinary duck ranch."

The authors point out that, in addition to cholera, two other diseases cause high mortality on the ranches—the so-called anatipestifer infection and an infectious disease which, though it has been identified, has not yet been described.

THE OCCURRENCE OF AVIAN TUBERCLE BACILLI IN DRESSED POULTRY

By W. H. FELDMAN

THIS article deals with the finding of avian tubercle bacilli in the spleens of dressed poultry in which no macroscopic evidence of tuberculosis was found in any organ. In all, the spleens from 121 fowls of different ages and including 18 ducks and 4 turkeys were examined. Cultural methods were used in an attempt to demonstrate the presence of tubercle bacilli and from the emulsions of the spleens yielding colonies of acid-fast bacilli, sub-cultures were made and tests were carried out for pathogenicity. The methods adopted are described and the result was that four spleens yielded tubercle bacilli of the avian type.

The author in his comment states: "It seems reasonable to assume that had material from more adult chickens been examined more positive results might have been secured. . . . However, the fact that tubercle bacilli were obtained from the tissues of fowls that were in good physical condition and without gross evidence of disease emphasises the difficulty of eliminating every tuberculous fowl even by competent methods of poultry inspection. Likewise these findings emphasise the importance of rearing poultry in a non-tuberculous environment if the ultimate consumer is to be assured that the dressed poultry he buys is not likely to be infected with tuberculosis."

Journ. Infect. Dis. (1938), LXIII, 332.

In the summary it is stated that the findings indicate that dressed poultry originating from districts where tuberculosis of fowls is prevalent may harbour avian tubercle bacilli of standard virulence, even though recognisable morbid changes be absent, that tubercle bacilli may be present in apparently normal fowls reared in an infective environment, and that no practical method of post-mortem inspection will disclose the presence of tubercle bacilli in the absence of gross lesions. The author suggests that the correction of the situation consists in rearing fowls in a non-tuberculous environment.

(Note.—No mention is made of the use of the Tuberculin Test.)

HISTOLOGICAL INVESTIGATIONS INTO THE NATURE OF SO-CALLED MAREK'S DISEASE (FOWL PARALYSIS)

By K. POTEL

THE author reviews some of the literature on fowl paralysis, especially that dealing with the nature of the lesions. He compares the points of view of the German School with those of the American and British workers and sets out by making histological examinations on the tissues of 32 affected fowls to come to a separate opinion. The gross lesions are discussed in each case, and it was noticed that in only 21 of the 32 cases were macroscopic lesions found in the nerves. On the other hand, however, in 11 cases some of the organs were obviously abnormal (heart, liver, kidneys, wall of intestine, lungs, spleen, ovary, etc.).

Full histological examinations of nervous and other tissues were made in all cases and the types of cells are fully discussed. The infiltrative processes are discussed and it is concluded that in all probability the cells typically found in affected nerves and other tissues (lymphocytes) are not the cells found in the early stages of the condition but follow on after a pathological process, e.g., degeneration of the early invading cells.

The result of the histological investigations lead to the following conclusions: (1) The cellular infiltrations of the central and peripheral nervous system have a close resemblance, morphologically, to the cellular formations in the lesions of the affected organs. (2) Primary degenerative changes in the nerves do not occur: the destruction of the nerve fibres is entirely due to pressure atrophy from cellular infiltration. (3) Concerning the cellular

Zeitschr. f. Infektionskr. parasit. Krankh. u. Hyg. d. Haust (1938), XLV, 141.

infiltrations, active cell division of the lining endothelial cells of the capillary and small blood vessels seem to play an essential part.

The author comes to the following conclusion: "From these considerations, together with the common occurrence of lymphomatic lesions in the various organs of birds affected with Marek's disease, I have come to the conclusion that Marek's disease (fowl paralysis) is not a polyneuritis nor a neuro-myelitis, but must be regarded as a form of fowl leucosis and should be referred to as neurolymphomatosis gallinarum."

News

THE POULTRY INDUSTRY IN 1938

By MAJOR IAN MacDOUGALL, O.B.E.
Secretary, National Poultry Council.

THE decline in the poultry population in Great Britain and Northern Ireland which commenced in 1935 was arrested in 1938, as is indicated by the census on June 4 last, i.e., on holdings above one acre in extent. The total increased by 0.34 per cent., but it is still 11.66 per cent. below the peak year, 1934. The number of fowls decreased by 0.05 per cent., and duck, geese and turkeys increased by 5.97 per cent. The following analysis indicates the increases and decreases, in percentages, in the various types of poultry. (A—over six months, B—under six months). No figures are available as to holdings of one acre and under.

	Fowls.		Turkeys.	
	A.	B.	A.	B.
England	—6.00	+4.33	—2.16	+18.63
Wales	—6.16	+6.70		
Scotland	—2.48	+7.64	+9.32	+28.30
N. Ireland	—4.71	+4.39	—2.91	+ 9.87

	Ducks.	Geese.
	A and B.	A and B.
England	+ 3.73	+11.75
Wales	—11.25	+ 3.62
	A. B.	A. B.
Scotland	+0.44 +15.21	—3.57 +12.38
N. Ireland	—8.55 +15.27	—7.81 + 4.44

It is regretted that, in the case of England and Wales, it is not possible to give separate figures of over and under six months for ducks and geese as the Ministry of Agriculture did not obtain separate figures in these age groups.

As in the previous year a supplementary census was taken on September 4 of turkeys on agricultural holdings in England and Wales. The estimated

number, at that date, was 948,000, compared with 788,515 on June 4, an increase of 20.23 per cent. A similar inquiry was conducted by the Department of Agricture for Scotland, and the increase over June 4 was 159,000, or 8.95 per cent. These increases consisted mainly of young turkeys hatched since June 4 and represented an additional supply for the Christmas trade.

Comparisons With Cereal Crops

As in previous annual summaries, I am able, by the courtesy of the respective agricultural departments, to give the estimated production values of grain compared with egg and poultry production during 1938 in Great Britain and Northern Ireland.

The production of wheat increased by 455,308 tons, of oats by 144,946 tons, and of barley by 246,784 tons. There were, however, decreases in the prices obtained. The value of wheat in 1938 decreased by £810,965, of oats by £1,008,440, and the value of barley increased by £1,973,717. The figures are as follows:—

ESTIMATED PRODUCTION VALUES OF CEREAL CROPS, 1938.

	Wheat.	Oats.	Barley.
England and Wales	£12,521,250	£8,106,583	£8,163,834
Scotland	667,333	4,325,500	873,833
N. Ireland	32,973	1,557,161	24,641
	£13,221,556	£13,989,244	£9,062,308

ESTIMATED PRODUCTION VALUE OF EGGS AND POULTRY, 1938.

England and Wales	£25,763,533
Scotland	3,806,263
N. Ireland	5,058,787
	£34,628,583

The total values of grain crops in Great Britain and Northern Ireland were £36,273,108, being greater than those for 1937 by £154,312.

The value of egg and poultry production in Great Britain and Northern Ireland was less than that of all cereal crops by £1,644,525.

It is of interest to note that in 1938 the value of eggs and poultry production in Great Britain and Northern Ireland is computed to be greater by £21,407,027 than that of wheat, by £20,639,339 than that of oats and by £25,566,275 than that of barley.

General Observations

As was indicated in the opening paragraph, the decline in poultry population which set in in 1935 was arrested in 1938. There was a decrease in the number of birds over six months of age, but there was an increase in the number under that age which more than offset that decrease. The price of wheat was considerably reduced, and there were slightly improved prices for eggs in the first six weeks of the year. These, in conjunction with favourable

rearing conditions, encouraged expansion. The early promise of better prices for eggs was not long maintained. Towards the end of February prices fell below the average for the previous year and continued until the end of April. From that point onwards slightly better prices were obtained and continued until October, when another fall in prices occurred. Undoubtedly prices were affected by the enormous increases in imports of eggs in shell and eggs not in shell. Both attained record proportions in 1938. The former amounted to 27,697,400 long hundreds (3,323,688,000 eggs), an increase of 2,955,700 long hundreds over 1937. The previous highest total imported was in 1930, when the amount was 26,541,280 long hundreds.

Imports of eggs not in shell in 1938 amounted to 944,209 cwt., an increase of 97,030 cwt. over 1937. Despite the war in the East, supplies from China only decreased by 28,309 cwt. (3.44 per cent.). Imports from other foreign countries increased by 120,682 cwt., an increase of 551.34 per cent. Other foreign countries were not slow to take advantage of the increased prices, due primarily, if not solely, to the hostilities in the East and the uncertainty caused thereby.

Under the Import Duties Act of 1932, these imports were subject to an *ad valorem* duty of 10 per cent. The Import Duties Advisory Committee are empowered, under Section 16 of the Finance Act of 1933, to recommend a variation in the rate of duty so that it shall continue to approximate 10 per cent. of the average import value, and in 1934 a specific rate was substituted. That Committee had under consideration the rise of values in 1938, but, owing to the unsettled conditions in the East, felt themselves unable to recommend alternative specific duties which could be depended upon to give an *ad valorem* incidence of 10 per cent. In the circumstances the Committee recommended that the duty should revert to 10 per cent. *ad valorem*. The effect of this alteration in the duty will be better appreciated when it is pointed out that the average value, per cwt., of liquid or frozen eggs in 1938 was £3 4s. 9¼d. The specific rate of duty was 4s. 8d. per cwt., whereas had the 10 per cent. *ad valorem* duty applied the charge would have been approximately 6s. 6d. per cwt. The Order giving effect to the alteration came into force on January 18, 1939.

Mortality still continues to have a substantial effect on the economic position of producers. In the 49 laying trials (open, county and Government controlled) officially recognised by the National Poultry Council in 1937-38, the average pullet mortality decreased by 0.78 per cent. The average mortality at these trials was 16.84 per cent., and this is the only source where definite figures can be obtained of a general average. Figures at the present 1938-39 trials do not indicate, at present, that there is likely to be any decrease in the average.

The Poultry Technical Committee of Great Britain issued their report in February, 1938. The Government were impressed by the Committee's views

as to the very serious and urgent nature of the disease problem in the poultry industry, and they agree substantially with the recommendations of the Committee as to the manner in which the problem should be tackled. It is proposed that a Poultry Commission for Great Britain shall be set up with, amongst other things, power to control the distribution of breeding stock, hatching eggs and day-old chicks.

Another factor contributing to the present difficulties of the industry is the lack of organisation in the marketing of home-produced eggs and poultry. In December, 1937, a Joint Committee of the National Poultry Council and the National Farmers' Union submitted, to the Minister of Agriculture, main principles on which they thought marketing organisation could be effected under the Agricultural Marketing Acts. These proposals were conditional on adequate regulation of imports and adequate financial assistance from the Government. The proposals were approved by large majorities of the affiliated bodies and branches of the two organisations. In announcing the Government's Long Term Policy for the Poultry Industry in the House of Commons on July 11, the Minister of Agriculture pointed out that such a large proportion of individual poultry-keepers were in a small way of business that the regulation of marketing under a producers' marketing scheme would present a very formidable task, and it was doubtful whether such a scheme would be the most effective, or, indeed, the most appropriate, machinery for securing, for example, the standardisation of grades, since this is an object which necessarily involves the regulation of marketing practices in the wholesale and retail distributive trades. As an alternative to action by the producers under the Agricultural Marketing Acts, the Government propose that the Poultry Commission should be given power to regulate the marketing of eggs, and also of dead poultry, by the standardisation of grades, etc., up to the point of final sale to the consumer. Organisation of marketing is to be given effect to through the control of distributive functions. In the Poultry Bill which is to give effect to the recommendations in regard to disease control and marketing organisation, it is also proposed to take power to control imports, without which any organisation would be wholly ineffective. The Bill is expected early in 1939. When it is passed into law it will take some time to set up the necessary administrative machinery. In the meantime, so far as the disease problem is concerned, breeders themselves can very materially assist in dealing with the question by a return, in so far as the present economic position will allow, to more natural methods of breeding and rearing.

Egg Imports

In the table opposite are given the figures from the Trade and Navigation Returns, published by the Board of Trade, for the two years 1937 and 1938, showing the quantities of eggs imported from the countries enumerated. As

usual, the percentages of increase or decrease in 1938 as compared with the previous year are shown.

QUANTITIES OF EGGS IN SHELL IMPORTED INTO GREAT BRITAIN AND NORTHERN IRELAND, 1937 AND 1938

Country of Origin	Great Hundreds		Percentages inc. or dec. 1938 from 1937
	1937	1938	
Eire	1,949,200	2,218,000	13.79 inc.
Union of South Africa	256,700	270,900	5.53 inc.
Australia	1,262,800	1,009,900	20.03 dec.
Other British Countries	152,300	175,400	15.17 inc.
Finland	501,900	517,500	3.11 inc.
Sweden	332,800	484,700	45.64 inc.
Denmark	9,997,700	9,511,000	4.87 dec.
Poland	2,256,800	2,621,100	16.14 inc.
Netherlands	4,309,600	5,932,900	37.67 inc.
Belgium	436,300	107,400	75.38 dec.
China	1,090,500	298,200	72.65 dec.
Other Foreign Countries	2,195,100	4,550,400	107.30 inc.
	24,741,700	27,697,400	11.95 inc.

Supplies from Empire sources accounted for 13.27 per cent. of the total imports, the balance of 86.73 per cent. coming from foreign sources. Imports from the former increased by 1.47 per cent. and from the latter by 13.74 per cent. Denmark's share of the total imports was 34.34 per cent. Supplies from the Netherlands have increased annually during the past four years, and their share in 1938 was 21.42 per cent. If the imports of eggs not in shell are translated into terms of units at the rate of 1,120 eggs to the hundredweight, they represent a total of 1,057,514,080 eggs, and if these are added to the imports of eggs in shell, the grand total of imports represents 4,381,202,080 eggs.

TOTAL VALUES OF EGGS IMPORTED IN SHELL, 1938, AND AVERAGE VALUES FOR 1937 AND 1938

Country of Origin	Total Values, 1938	Values per Great Hundred, 1937	1938
	£	s. d.	s. d.
Eire	994,570	6 10¼	8 11½
Union of South Africa	156,714	11 8¼	11 6¾
Australia	599,991	11 5	11 10½
Other British Countries	102,768	11 7¼	11 8½
Finland	234,516	8 8½	9 0¾
Sweden	207,556	8 6½	8 6¾
Denmark	4,643,118	8 9½	9 9¼
Poland	878,439	6 2½	6 8½
Netherlands	2,699,408	9 0¼	9 1¼
Belgium	51,280	9 3¼	9 6½
China	114,449	7 3¼	7 8
Other Foreign Countries	1,691,066	7 5¼	7 5¼
	12,373,875	8 5¼	8 11¼

The increase in total value of eggs in shell imported over 1937 was £1,930,621, or 18.49 per cent., and the increase in average values was 6d. per great hundred.

EGGS NOT IN SHELL
(Liquid or Frozen—Whole, Yolk or White)

Country of Origin	Hundredweights		Percentages inc. or dec. 1938 from 1937
	1937	1938	
British Countries	2,636	7,293	176.67 inc.
China	822,654	794,345	3.44 dec.
Other Foreign Countries	21,889	142,571	551.34 inc.
	847,179	944,209	11.45 inc.

In 1937 China's share of these imports was 97.22 per cent. In 1938 this had decreased to 84.13 per cent., and supplies from other foreign countries accounted for 15.10 per cent. During 1937 and 1938 the value of these imports has steadily increased. In 1936 the average value, per cwt., was £2 8s. 6d., in 1937 £2 11s. 5½d., and in 1938 £3 4s. 9½d. The following table indicates the development of the export of liquid or frozen eggs by other foreign countries consequent upon the decline of supplies from China.

Country of Origin	Hundredweights	
	1938	1937
Australia	6,408	812
Canada	873	1,805
Eire	12	19
China	794,345	822,654
Japan	19,277	5,662
United States of America ...	8,392	750
Argentina	680	—
Brazil	121	241
Hungary	35,876	1
Roumania	23,477	428
Poland	12,121	—
Yugoslavia	16,413	—
Netherlands	4,867	677
Latvia	4,080	—
Denmark	3,465	2,187
Italy	1,450	—
Belgium	372	328
Germany	279	255
France	19	27
Egypt	11,682	11,333
	944,209	847,179

Each year there is a re-export of eggs not in shell which reduces the above totals. In 1938 these were 50,192 cwt.—in value £154,198—representing 5.32 per cent. of the volume of this class of imports.

Imports of dried eggs, whole, yolk and albumen, amounted in 1937 to

33,688 cwt., and in 1938 to 29,616 cwt., a decrease of 4,072 cwt. Their figures are included in the net value of imports but are not included in the estimate of consumption values, as it is not possible to say what quantity of these are used for manufacturing purposes. The average value, per cwt., of these products in 1936 was £9 12s. 5½d., in 1937 £9 19s., and in 1938 £11 13s. 5½d.

IMPORTS OF DEAD DOMESTIC POULTRY

Country of Origin	Quantities cwts.	Values £
Eire	101,989	458,915
Other British Countries	15,182	87,806
Soviet Union	5,579	17,426
Netherlands	24,427	110,034
Austria	447	1,862
Hungary	173,920	620,610
Yugoslavia	43,296	141,856
Other Foreign Countries	80,643	308,908
	445,483	1,747,417

Imports decreased by 60,405 cwt., or 11.94 per cent., and values by £233,424, or 11.78 per cent.

NET VALUES OF IMPORTS, 1938

	£
Eggs in shell	12,373,875
Eggs not in shell	3,403,825
Dead poultry	1,747,417
	17,525,117

The value of total imports in 1938 was £2,586,133 higher than those for 1937.

An estimate as to value of total production and consumption of eggs and poultry in Great Britain and Northern Ireland, and adding imports, is as follows:

	£
Home production	34,628,583
Imports of eggs and poultry, inclusive of eggs not in shell, but exclusive of dried eggs and re-exports	17,025,216
	51,653,799

Of the total values as stated, 67.04 per cent. represented home production and 32.96 per cent. came from Empire and foreign countries.

THE VALUE OF COD-LIVER OIL AS A SUPPLEMENTAL FOOD FOR CATTLE AND POULTRY

COD-LIVER OIL has a reputation in veterinary practice as old and as deserved as it has in medical practice, and the history of the oil from the point of view of both professions has many similarities. From fifty to a hundred years ago, with the development of indoor methods for keeping livestock, cod-liver oil was regularly used, and a drum of cod-liver oil in those days was a normal item in the ordinary farm's medicine chest just as it was a normal item in the ordinary household. It was especially intended in both cases for the needs of the young and also for use with adults when their state of health suggested that they might benefit.

In farming practice the old practice with cod-liver oil was to give it in a special mash once or twice a week to cattle and horses during the winter season. In these times neither poultry, pigs, nor even dairy cattle were major or even important elements in the winter livestock population of the country, nor was any serious effort made to keep them in full production during the winter time.

The old use of cod-liver oil as a special winter-time food for horses and cattle suffered a considerable decline, first of all by substitution of the oil—which was relatively expensive—by cheaper forms of fish oil such as seal oil, and finally by the use of linseed oil. But even the linseed oil mash has, for a variety of reasons, itself suffered a decline, though it is still used widely in feeding practice and as a medicine.

In medical practice there was a corresponding change-over from cod-liver oil to olive oil. These changes, to some extent, arose through a confusion of ideas or rather through a lack of knowledge. The brilliant researches of Sir Gowland Hopkins and Sir Edward Mellanby some twenty or thirty years ago made it quite clear that cod-liver oil possesses virtues and feeding values which neither seal oil, olive oil nor linseed oil show. These feeding values arise from the fat soluble vitamins A and D which cod-liver oil contains in abundance.

The whole modern development of livestock farming has made it more than ever essential that there should be an effective return to the old custom of feeding cod-liver oil regularly either as a standard ingredient used in the food or, alternatively, as rich special mashes or feeds given systematically during the winter months to all classes of livestock, particularly to poultry, pigs, dairy cattle, horses and to all forms of young livestock. Further than this, the need is greatest for livestock kept indoors and fed on concentrated foods. The reasons for this are now quite clear. The food which animals obtain out of doors, and especially herbivorous animals which are naturally

equipped to deal satisfactorily with large amounts of grass, is outstandingly rich in vitamin values, and especially vitamin A or its precursor carotene. The foods which are fed to animals indoors are predominantly poor in vitamin values, and especially in vitamin A values. In fact, the only food fed to animals indoors (apart from such green food as it is found possible to feed to them) that contains a measurable amount of vitamin A is yellow maize, which contains rather less than a 200th part of the amount of vitamin A found in good cod-liver oil. No indoor fed food contains measurable amounts of vitamin D.

There is now little doubt that the spectacular improvement in the general health and well-being of stock when they are turned out to fresh young grass in the springtime arises very largely from the abundant vitamin values, and especially vitamin A values, which the grass provides as compared with the outstanding vitamin A poverty of the winter feed given to them before they are turned out. There is also the additional benefit from direct sunlight, which can of itself supply vitamin D.

Nutrition and Disease

Most of the information which we now have as to the inter-relationships between nutrition and disease has arisen from work on experimental animals —rats, dogs, chickens and pigs. It is nevertheless remarkable that while these lessons are being brought home to our human population and are, in fact, the basis of the modern drive against malnutrition and its diseases, which characterises national and international efforts to improve human health, these same lessons are not being applied to anything like the same extent in veterinary practice, though in some spheres of farm feeding cod-liver oil has certainly secured for itself substantial use in this country. For example, the present consumption of cod-liver oil as a feeding stuff, mainly for poultry, exceeds 6,000 tons per annum; but even this quantity is but a fraction of what is really necessary to keep poultry alone in health under modern conditions of production. An outstanding instance of failure to appreciate the relationship between nutrition and disease, even in the poultry industry, is that the recent poultry commission which studied the whole state of the poultry industry, particularly the appalling incidences of disease, barely mentioned the question of nutrition in relation to the mortality rate. There is every reason to think that failure to appreciate the finer points of nutrition and the needs of livestock in relation to it is responsible for many of the conditions which the report sets out.

It is with those classes of livestock—poultry, pigs and dairy cattle in heavy winter production—that disease is most widespread and exacts the heaviest toll. It is further with these classes of livestock that diseases tend to become mysterious both as to origin and cause. It has often been the habit in farming and veterinary practice to "blame the food" when no

other apparent explanation could be found for casualties and disease. In the light of the knowledge hitherto available such blame seldom could be justified since, judged by the ordinary standards of analysis and composition, no real cause of trouble could usually be made clear. Since, however, the existence of accessory food factors as necessary components of food became recognised, it is now clear that unless these accessory food factors are provided, foods which may appear to be complete may be seriously lacking in the essentials necessary for proper nutrition.

Under modern conditions, farming livestock has been industrialised, just as our human population was industrialised some hundred years ago. It is significant that, just as cod-liver oil found its way into medical practice with the commencement of our industrial life, so it is equally becoming important in farming practice with the industrialisation of our livestock. The reason for this is quite clear. With the sole exception of yellow maize, all purchased and home-grown foods that are used for hand feeding to livestock are predominantly deficient—indeed, to all intents and purposes devoid—either of vitamin A or vitamin D value. The outstanding difference between a food that animals obtain for themselves under so-called natural conditions out of doors, and the food which the farmer gives them, lies in this matter of richness in vitamin value.

The true function of cod-liver oil as a supplementary food, and as something which will ward off diseases arising from deficiency, is the abundant presence in it of the two fat soluble vitamins, coupled with the fact that the oil itself, if presented as it should be in an undamaged condition, is an essential feeding stuff and provides the animal with a supply of metabolic fat of the type essential to life and of the type which an animal cannot easily create for itself either from carbohydrate, or from either pre-existing fats of different molecular weight and lower degree of unsaturation.

Reviews

Hobday's Surgical Diseases of the Dog and Cat. Fourth Edition by JAMES MCCUNN, M.R.C.V.S., M.R.C.S., L.R.C.P. (Professor of Veterinary Surgery, Royal Veterinary College, London). Pp. vii + 398. 311 Illustrations. Price 21s. net. Postage 6d. Abroad 1s. London: Baillière, Tindall & Cox.

The publication of a new edition of Hobday's "The Surgical Diseases of the Dog and Cat" is always a welcome event. It now enters on its fourth

edition, and is edited by Professor J. McCunn, Professor of Anatomy, Royal Veterinary College, London: in the absence of its original author it would be hard to find one more fitted to take his place.

Professor McCunn mentions that Hobday placed canine and feline surgery in its proper sphere. He did. The writer watched some of his efforts as a student, and knows that our position as canine surgeons is due to what he did in College then, his teaching, and the knowledge stored in the little green book which one sees on the shelf in almost every surgery.

We now have it brought up to date by the inclusion of developments in anæsthesia, minor and major operations. Among anæsthetics perhaps one would have liked to read more about nembutal, but sufficient is said to ensure confidence in its increasing use. After all, surgical knowledge is not gained by reading but by performing. The methods of operating for the various conditions met with in such organs as the eye, ear, etc., are clearly described and abdominal operations, some made more frequent by the showings of X-rays, are dealt with by various methods to be chosen as the surgeon fancies. Ovariotomy in both dog and cat still remains more or less a groping in the dark performance, though in the cat and possibly in the bitch it need not be. We are usually afraid of making abdominal incisions too large and often do not make them large enough.

Under laparotomy we are told that a protective bandage is often unnecessary and a source of irritation. It may be, but in the only case in which the writer neglected to apply one the intestine prolapsed a week after.

The treatment of fractures is very well dealt with and will be welcomed owing to the numerous cases now met with on the road and race track. Many improved methods of dealing with them are shown.

A special chapter is devoted to the application of radium by Mr. Cameron Wright, but one would think its cost and uncertainty of action will preclude its use in our animals.

The new edition of this book is full of interesting matter, helpful to both student and practitioner. In fact, as a standard work we cannot do without it, for it is as valuable in surgery as it is in diagnosis. Professor McCunn is to be congratulated on the way he has performed his task and on the inclusion of many helpful illustrations.

Principles of Veterinary Science. By FREDERICK BROWN HADLEY, D.V.M., Professor of Veterinary Science in the University of Wisconsin, and Veterinarian of the Wisconsin Agricultural Experiment Station. Third Edition. Entirely reset. Pp. 594. 126 Illustrations. London: W. B. Saunders Company, Ltd. Price 18s. net.

The preface states that it is a book written for the student of agriculture, presenting veterinary science in its relations with natural and physical sciences;

and one might add commerce, for the by-products of the abattoir are mentioned—very little has been left out.

Here we have a synopsis of anatomy, physiology, histology, morphology, surgery, medicine, bacteriology, toxicology, endocrinology, biochemistry, etc., all in one cover, and intended for a farm student, very few of whom, in this country, have had a proper start-off in simple chemistry, botany and physics.

Evidently in the mind of the author it is the prevailing idea for agricultural education, as in other branches, to "stuff it in" and chance the consequences, which are so often unfortunately confusion.

It is difficult to say how far such a book will aid a student. But of the vast array of matter—594 pages—he or she may be able to pick out information which will be of use in later days; but it does seem a lot for a student of agriculture to digest.

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